Set	Items	Description
S1	418	AU="GLENN J"
S2	56	AU="GLENN J."
S3	37	AU="GLENN JEFF" OR AU="GLENN JEFFERY" OR AU="GLENN JEFFREY" OR AU="GLENN JEFFREY K" OR AU="GLENN JEFFREY S" OR AU="GLENN JERRY L"
S4	511	S1 OR S2 OR S3
S5	22033	PRENYL?
S6	6646294	PROTEIN
S7	8	S4 AND S5 AND S6
S8	1	S7 NOT PY>1993
S9	2623442	VIRUS OR VIRAL
S10	2376	S5 AND S6 AND S9
S11	11635247	TREAT?
S12	5922527	INHIBIT?
S13	251202	ANTIVIRAL
S14	16296802	S11 OR S12 OR S13
S15	2284	S10 AND S14
S16	720	S5(S)S6(S)S9
S17	16	S16 NOT PY>1993
S18	8	RD (unique items)
S19	184	MIMIC(S)S5
S20	822	PRENYL(W)TRANSFERASE
S21	11475	MEVALONATE
S22	174	S19 AND S14
S23	102	S22 AND S9
S24	1	S23 NOT PY>1993
S25	1	RD (unique items)
S26	124	S9 AND S14 AND S20
S27	4	S26 NOT PY>1993
S28	4	RD (unique items)
S29	322	S9 AND S14 AND S21
S30	69	S29 NOT PY>1993
S31	58	RD (unique items)

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18/3/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07828438 94047387
Isoprenylation mediates direct protein-protein interactions between hepatitis large delta antigen and hepatitis B virus surface antigen.
Hwang SB; Lai MM
Howard Hughes Medical Institute, University of Southern California School of Medicine, Los Angeles 90033-1054.
Journal of virology (UNITED STATES) Dec 1993, 67 (12) p7659-62, ISSN 0022-538X Journal Code: KCV
Contract/Grant No.: AI 26741, AI, NIAID
Languages: ENGLISH
Document type: JOURNAL ARTICLE

18/3/5 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

0159466 DBA Accession No.: 94-02017 PATENT
Inhibiting virion morphogenesis, production, release or uncoating- hepatitis A virus, hepatitis C virus, hepatitis D virus, herpes simplex virus, cytomegalo virus, varicella-zoster virus, influenza virus, etc. virus inactivation
PATENT ASSIGNEE: Univ.California 1993
PATENT NUMBER: WO 9324660 PATENT DATE: 931209 WPI ACCESSION NO.: 93-405848 (9350)
PRIORITY APPLIC. NO.: US 890754 APPLIC. DATE: 920529
NATIONAL APPLIC. NO.: WO 93US5247 APPLIC. DATE: 930601
LANGUAGE: English

18/3/6 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120069589 CA: 120(7)69589n PATENT
Method using inhibitors of prenylation and of post-prenylation reactions for inhibition of viral morphogenesis, production, release, or uncoating
INVENTOR(AUTHOR): Glenn, Jeffrey
LOCATION: USA
ASSIGNEE: University of California
PATENT: PCT International ; WO 9324660 A1 DATE: 931209
APPLICATION: WO 93US5247 (930601) *US 890754 (920529)
PAGES: 26 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12Q-001/70A; C12N-007/04B; C12N-007/06B; A61K-035/76B DESIGNATED COUNTRIES: AT; AU; BB; BG; BR; CA; CH; CZ; DE; DK; ES; FI; GB; HU; JP; KP; KR; KZ; LK; LU; MG; MN; MW; NL; NO; NZ; PL; PT; RO; RU; SD; SE; SK; UA; US; VN
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG

18/3/8 (Item 2 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00337738
METHOD FOR INHIBITION OF VIRAL MORPHOGENESIS
PROCEDE D'INHIBITION DE LA MORPHOGENESE VIRALE

28/3,AB/4 (Item 2 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00285961
METHODS AND COMPOSITIONS FOR THE IDENTIFICATION, CHARACTERIZATION AND INHIBITION **OF FARNESYL PROTEIN TRANSFERASE**
PROCEDES ET COMPOSITIONS SERVANT A L'IDENTIFICATION, A LA CARACTERISATION ET A L' INHIBITION **DE LA TRANSFERASE DE PROTEINE FARNESYLE**
Patent Applicant/Assignee:
BOARD OF REGENTS THE UNIVERSITY OF TEXAS SYSTEM
BROWN Michael S
GOLDSTEIN Joseph L
REISS Yuval
Inventor(s):
BROWN Michael S
GOLDSTEIN Joseph L
REISS Yuval
Patent and Priority Information (Country, Number, Date):
Patent: WO 9116340 A1 19911031
Application: WO 91US2650 19910418 (PCT/WO US9102650)
Priority Application: US 90510706 19900418; US 90615715 19901120
Designated States: AT AT AU BB BE BF BG BJ BR CA CF CG CH CH CM DE DE DK DK ES ES FI FR GA GR HU IT JP KP KR LK LU LU MC MG ML MR MW NL NL NO PL RO SD SE SE SN SU US US
Publication Language: English
Fulltext Word Count: 17803

English Abstract
Disclosed are methods and compositions for the identification, characterization and **inhibition** of farnesyl protein transferases, enzymes involved in the farnesylation of various cellular proteins, including cancer related ras proteins such as p21ras. One farnesyl protein transferase which is disclosed herein exhibits a molecular weight of between about 70,000 and about 100,000 upon gel exclusion chromatography. The enzyme appears to comprise one or two subunits of approximately 50 kDa each. Methods are disclosed for assay and purification of the enzyme, as well as procedures for using the purified enzyme in screening protocols for the identification of possible anticancer agents which **inhibit** the enzyme and thereby prevent expression of proteins such as p21ras. Also disclosed is a family of compounds which act either as false substrates for the enzyme or as pure **inhibitors** and can therefore be employed for **inhibition** of the enzyme. The most potent **inhibitors** are ones in which phenylalanine occurs at the third position of a tetrapeptide whose amino terminus is cysteine.

French Abstract
Procedes et compositions pour l'identification, la caracterisation et l' **inhibition** de transferase de la proteine farnesyle, enzymes servant a la farnesylation de diverses proteines cellulaires, y compris des proteines ras associees au cancer telles que la p21ras. On presente une tranferase de proteine farnesyle qui a un poids moleculaire entre environ 70000 et environ 100000 d'apres la chromatographie par exclusion de gel. L'enzyme semble comprendre une ou deux sous-unites d'environ 50 kDa chacune. On decrit aussi des procedes d'analyse et de purification de l'enzyme, ainsi que des procedures d'utilisation de l'enzyme purifiee dans des protocoles de triage pour l'identification d'eventuels agents anticancereux qui inhibent l'enzyme et empechent ainsi l'expression de proteines telles que la p21ras. On presente enfin une famille de composes qui agissent soit comme de faux substrats pour l'enzyme ou comme **inhibiteurs** purs et peuvent par consequent etre employes pour l'**inhibition** de l'enzyme. Les

31/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07478198 93054571
Isoprenoid requirement for intracellular transport and processing of murine leukemia virus **envelope protein.**
Overmeyer JH; Maltese WA
Weis Center for Research, Geisinger Clinic, Danville, Pennsylvania 17822.
Journal of biological chemistry (UNITED STATES) Nov 5 1992, 267 (31) p22686-92, ISSN 0021-9258 Journal Code: HIV
Contract/Grant No.: CA34569, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Lovastatin blocks the biosynthesis of the isoprenoid precursor, **mevalonate** . When Friend murine erythroleukemia (MEL) cells are cultured in medium containing lovastatin, the precursor of murine leukemia **virus** envelope glycoprotein (gPr90env) fails to undergo proteolytic processing, which normally occurs in the Golgi complex. Consequently, newly synthesized envelope proteins are not incorporated into **viral** particles that are shed into the culture medium. gPr90env appears to be localized in a pre-Golgi membrane compartment, based on its enrichment in subcellular fractions containing NADPH-cytochrome c reductase activity and the sensitivity of its carbohydrate chains to digestion with endoglycosidase H. Arrest of gPr90env processing occurs at concentrations of lovastatin that are not cytostatic, and the effect of the **inhibitor** is prevented by addition of **mevalonate** to the medium. The low molecular mass GTP-binding proteins, rab1p and rab6p, which are believed to function in early steps of the exocytic pathway, are normally modified posttranslationally by geranylgeranyl isoprenoids. However, in MEL cells **treated** with 1 microM lovastatin, nonisoprenylated forms of these proteins accumulate in the cytosol prior to arrest of gPr90env processing. These observations suggest that lovastatin may prevent **viral** envelope precursors from reaching the Golgi compartment by blocking the isoprenylation of rab proteins required for ER to Golgi transport.

31/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05770114 90002911
Abolition of mevinolin-induced growth inhibition **in human fibroblasts following transformation by simian** virus **40.**
Larsson O; Barrios C; Latham C; Ruiz J; Zetterberg A; Zickert P; Wejde J
Department of Tumor Pathology, Karolinska Institutet, Stockholm, Sweden.
Cancer research (UNITED STATES) Oct 15 1989, 49 (20) p5605-10, ISSN 0008-5472 Journal Code: CNF
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The basal level of the gene expression and the activity of 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase was higher in SV40-transformed human fibroblasts (90-VA VI) than in normal ones (HDF). In both these cell types mevinolin (25 microM) caused an 85-90% depression of HMG CoA reductase activity and of the incorporation of [3H]acetate into sterols. In HDF this was coupled to an efficient block of cell growth, whereas the growth of 90-VA VI was only slightly reduced by mevinolin. In HDF, mevinolin (25 microM) also abolished essentially all dilichol synthesis, as measured by incorporation of [3H]acetate. In contrast, dolichol synthesis remained unaltered, or was increased, in mevinolin-

Patent Applicant/Assignee:
  THE REGENTS OF THE UNIVERSITY OF CALIFORNIA
  GLENN Jeffrey
Inventor(s):
  GLENN Jeffrey
Patent and Priority Information (Country, Number, Date):
  Patent: WO 9324660 A1 19931209
  Application: WO 93US5247 19930601 (PCT/WO US9305247)
  Priority Application: US 92890754 19920529
Designated States: AT AU BB BG BR CA CH CZ DE DK ES FI GB HU JP KP KR KZ LK
  LU MG MN MW NL PL PT RO RU SD SE SK UA US VN AT BE CH DE DK ES FR GB GR
  IE IT LU MC NL BF BJ CF CG CI CM GA GN ML MR NE SN TD TG
Publication Language: English
Fulltext Word Count: 5336
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inhibiteurs les plus puissants sont ceux dans lesquels la phenylalanine apparait a la troisieme position d'un tetrapeptide dont la terminaison amino est la cysteine.

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treated 90-VA VI. We suggest that these different responses of dolichol synthesis may depend on different substrate affinities of the rate-limiting enzyme in the dolichol pathway. However, if 90-VA VI was **treated** with 25-hydroxycholesterol (25-OH), an alternative **inhibitor** of HMG CoA reductase, the cellular growth as well as dolichol synthesis was significantly decreased. Since the **inhibitory** effect of 25OH on HMG CoA reductase activity did not exceed that of mevinolin, it seems that 25-OH, besides HMG CoA reductase, **inhibits** steps distal to HMG CoA reductase. This notion was further supported by the finding that addition of **mevalonate** did not prevent the 25-OH-induced growth **inhibition** . However, if dolichol was added along with 25-OH, the block was partially prevented, indicating that a critical level of de novo synthesis of dolichol for cellular growth.

31/3,AB/26 (Item 20 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01184600 Genuine Article#: GC831 Number of References: 64
Title: CELL-CYCLE-DEPENDENT, DIFFERENTIAL PRENYLATION OF PROTEINS (Abstract Available)
Author(s): SEPPLORENZINO L; RAO S; COLEMAN PS
Corporate Source: NYU,DEPT BIOL,BIOCHEM LAB/NEW YORK//NY/10003; NYU,DEPT BIOL,BIOCHEM LAB/NEW YORK//NY/10003
Journal: EUROPEAN JOURNAL OF BIOCHEMISTRY, 1991, V200, N2, P579-590
Language: ENGLISH Document Type: ARTICLE
Abstract: Isoprenylated proteins related to cell growth have been detected during proliferation. Since cholesterogenesis (isoprenoid synthesis) is mandatory for cell proliferation, the observation of a temporally coordinated protein prenylation during the cell division cycle might constitute obligatory processes in the signalling pathway for initiating DNA replication and/or in maintaining the growing state. We have found such a definitive cell-cycle-phase-dependent pattern of prenylation for various classes of cytosolic and nuclear matrix proteins in synchronized HepG2 cells. Characteristic [H-3]**mevalonate** incorporation began to increase during mid-to-late G1, just after cholesterol synthesis reached its apex, and peaked just prior to or coincident with mid S. Incorporation then declined subsequent to S (during G2) as cells approached mitosis. Prior to the rise in **mevalonate** incorporation into proteins, during early-to-mid G1, steady-state [C-14]acetate incorporation into chromatographically resolved cholesterogenic lipid intermediates displayed a maximum only into cholesterol. However, during the late-G1/S interval, a singular peak of C-14 incorporation was found for the farnesyl moiety (farnesol/nerolidol plus farnesyl diphosphate). Except for the farnesyl moiety, none of the other polyisoprenoids detected by our procedures showed any fluctuation in C-14 incorporation subsequent to mid G1. These results support the proposal that subsequent to peak cholesterol synthesis in early-to-mid G1, the generation of a cholesterol-pathway-dependent set of post-translationally modified, polyisoprenylated proteins could constitute an obligatory step leading to the duplication of the cellular genome, thereby impelling transit through the cell cycle. The well known high flux through cholesterogenesis in tumors, which manifests an intrinsic lack of sensitivity to feedback **inhibition** and operates continuously, is consonant with this proposal.

31/3,AB/38 (Item 1 from file: 76)
DIALOG(R)File 76:Life Sciences Collection

01722796 3012240

Isoprenoid requirement for intracelluar transport and processing of murine leukemia virus **envelope protein.**

Overmeyer, J.H.; Maltese, W.A.
Weis Center Res., Geisinger Clin., 100 N. Academy Ave., Danville, PA 17822-2616, USA

J. BIOL. CHEM. vol. 267, no. 31, pp. 22686-22692 (1992.)
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Virology Abstracts

Lovastatin blocks the biosynthesis of the isoprenoid precursor, **mevalonate** . When Friend murine erythroleukemia (MEL) cells are cultured in medium containing lovastatin, the precursor of murine leukemia **virus** envelope glycoprotein (gPR90 super(env)) fails to undergo proteolytic processing, which normally occurs in the Golgi complex. Consequently, newly synthesized envelope proteins are not incorporated into **viral** particles that are shed into the culture medium. gPr90 super(env) appears to be localized in a pre-Golgi membrane compartment, based on its enrichment in subcellular fractions containing NADPH-cytochrome c reductase activity and the sensitivity of its carbohydrate chains to digestion with endoglycosidase H. Arrest of gPR90 super(env) processing occurs at concentrations of lovastatin that are not cytostatic, and the effect of the **inhibitor** is prevented by addition of **mevalonate** to the medium. The low molecular mass GTP-binding proteins, rab1p and rab6p, which are believed to function in early steps of the exocytic pathway, are normally modified posttranslationally by geranylgeranyl isoprenoids. However, in MEL cells **treated** with mu M lovastatin, nonisoprenylated forms of these proteins accumulate in the cytosol prior to arrest of gPr90 super(env) processing. These observations suggest that lovastatin may prevent **viral** envelope precursors from reaching the Golgi compartment by blocking the isoprenylation of rab proteins required for ER to Golgi transport.
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